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A BRIEF ACCOUNT OF THE MICROSCOPICAL ANATOMY IN A CASE OF CHROME LEAD POISONING.

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This, as is well known, is not a rare disease, and the clinical phenomena of many cases have been frequently observed and recorded with every care, but the morbid anatomy of the various organs affected by the poison has only been investigated in a very limited number of cases, and in all, I believe, only about eighteen in number, the specific phenoměna of lead paralysis were far advanced; in some the muscles not only of the upper but of the lower extremities had suffered extreme atrophy during life, added to which the supervention before death in most cases, of eclampsia and other symptoms of severe implication of the central nervous system marked the majority as of established chronicity. The case from which I made some of the slides differs from all those hitherto recorded with the exception of one, in which the symptoms appear almost identical, inasmuch as the death of the patient occurred within six weeks subsequent to the supervention of one of the earliest symptoms of lead palsy, viz., the dropped wrist, though she had been suffering for some time from the effects of lead. The patient, a woman aged 48, had shown signs of unilateral peripheric paralysis for some two months before death. Married to a plumber, brass founder, etc., both Germans by birth, she lived in the rooms adjoining the front store, which was the plumber's shop and contained the usual outfit necessary for his trade. In the room off this store, to one side, was a store-room filled with lead pipes, paints, and sheets of lead for plumbing houses, etc. In this way the woman, who constantly helped her husband in the shop had been exposed to the influences of lead for some ten or fifteen years. The husband presented a weakly constitution, but only slight symptoms of any lead poisoning, the chief being colicky pains, but only at long intervals, which he laid to cold and long stooping. The woman told me she had suffered constantly from colic, which had laid her up for some days. About three months previous to the first visit she had been obliged to sit still and do nothing on account of constant sick headache, giddiness, and the stiffness of the extensor muscles of the right and left forearms, the left leg being slightly affected also. The general symptoms were great uræmia, colicky abdominal pains, complete loss of power in the right forearm, and some weakness and trembling in the left, severe burning headache, occasional emesis, and she said, "I feel very, very ill." The mouth showed signs of disease, the lips being cracked, very sore, and blistered, the tongue coated, tremulous, the gums tumid, and showed distinctly the characteristic sign—blue lines. The right wrist was "dropped," extension of hand and fingers impossible, the left arm only being slightly affected. The muscles were not much wasted, but soft and flabby. The left leg was slightly affected, œdematous. The abdominal walls were retracted, lungs and respiration normal, heart-sounds loud, normal area of heart dullness a little increased, distinct cardiac hypertrophy noted, the urine pale (renal secretion), low specific gravity, much albumen. At the next visit she was comatose, had repeated convulsive fits, several of which I witnessed. Death occurred the same day. Cause of death—primary, lead poisoning; secondary, uræmia. I may state here I knew of a case of lead poisoning occurring in a typesetter from handling it in his mouth. This patient went insane. Unfortunately the interest of this examination was spoiled by the difficulties under which an autopsy was granted, rendering the case but fairly complete. However, we succeeded in obtaining a piece of a nerve—musculo-spiral—right extensor digitorum communis muscle, which, alongside the supinator longus, was pale, resembling fish muscle; the spinal cord showed no abnormalities. The left kidney was obtained by nephrectomy, any other dissection being forbidden us. The microscopical examination of the *muscle* was examined fresh, then some stained with logwood. The fibers, most of which showed normal striation, were here and there slightly cloudy and enlarged, splitting longitudinally into fibrillæ, or the sarcolemma appeared to break up into spindle-shaped bits of all sizes; transverse striations still shown by most of the fibers, but some fibers show swellings and curvatures; but even those least altered show proliferation of the nuclei, almost all of which appear enlarged, and, with an immersion one-twelfth Zeiss, possess several granular bodies like nucleoli. All these different stages of atrophy can be seen in one and the same preparation. Side by side with the normal striated fibers are some which appear to break up longitudinally, while others are cloudy and granular. If these changes in the muscle be regarded as a whole, they will be found identical almost in the case of progressive muscular atrophy described by Friederich (14). The *nerve* was stained by Osmic acid and longitudinal sections made. The chief point was varicosity of the medullated substance, which appeared broken into globular and irregular masses on comparison with a healthy nerve treated in the same manner. It appeared also as if Ranvier's (5) nodes were more than usually distinct. Logwood longitudinal sections showed enormous proliferation of ovoid nuclei; in cross-sections the medullary substance appeared to be lost in many places, the connective tissue increased and development of nuclei. *The kidney weighed* 6½ ounces; had a slightly adherent capsule, and it showed a few retention cysts, small in size; cortical diameter was distinctly diminished. The microscope showed that the Malpighian corpuscles were greatly changed, showing in some only a proliferation of the nuclei and thickening of their capsule; others were completely degenerated and welded into homogeneous masses, almost hyaline in character. The convoluted tubules were more or less atrophied; their epithelium cloudy; laminæ studded with tufts of needle-like crystals of uric acid. The epithelium of the straight tubes also showed cloudy degeneration. The interstitial connective tissue was increased; the vessels were much thickened, and showed endoarteritic changes. In short, the kidney showed chronic interstitial nephritis, in which a large amount of uric-acid crystals present in the tubes appeared clearly shown. The sections were stained logwood and carmine and restained in logwood and mounted again, as they met with an accident during removing and are not so clear as formerly.

A portion of muscle was hardened in bichromate of ammonia solution; here again the proliferation of nuclei were distinctly seen, and here and there degeneration was visible, the main alterations being in the intramuscular nerve fibers, the connective tissue of these being much thickened and many nuclei present. All the primitive nerve fibers appeared to have a thickened sheath; in some the axis cylinder was distinctly visible, while logwood and carmine staining only showed a homogeneous mass. Nuclei were largely developed between the single nerve fibers. It was a matter of regret that the examination was so incomplete and the clinical features of the nephritis not studied so carefully as one could wish, for it seems that nephritis from lead poisoning is likely to throw a good deal of light on the pathology of Bright's disease. From the examination made it is evident the patient suffered from interstitial nephritis. There was, however, also a distinct cloudy degeneration of the epithelial cells in the contorted tubules which hardly takes the logwood stain, and a similar change in the cells of the straight tubules. It was, however, seen that in many places the altered epithelium was found in the immediate neighborhood of the impacted uric-acid crystals, and it is therefore highly probable that the altered condition of the epithelium, especially as it was found also in the collecting tubes, where a number of these crystals were also found, was a secondary effect and due to irritation set up by these bodies. To realize the enormous change produced in this lesion, a transverse section of the papillary portion of a diseased kidney should be compared with the same part of a normal one. The chief feature of importance is the abnormal condition of the arteries in chronic interstitial nephritis, the intima being many times thicker than the media. In the normal artery there is a very small amount of fibrous connective tissue lying between the fenestrated membrane of Henle and the endothelial lining of the vessel. The fibrous tissue is subjected to the same influence in the circulating blood that sets up the chronic inflammatory change throughout the kidney, and the effect produced is the same; it increases and grows inward, carrying the endothelial lining with it. In this way the lumen of the artery is narrowed and its caliber diminished. The muscular coat is not altered in any way. The process is one of chronic endarteritis, brought about by the chronic irritation produced by irritating substances in the blood. This change affects the arteries of the whole body and not the kidney alone. For example, the basilar artery will often show as marked changes as any artery in the kidney itself. The process by which the change has been set up is a gradual one, and the effect is a very gradual diminution in the caliber of the arteries of the systemic circulation. The force required to drive the same amount of blood through the narrowing arteries requires augmentation, the left side of the heart hypertrophies to overcome the increased resistance to the passage of the blood through the narrowed vessels. The adventitia of the arteries is unchanged. *Of the various theories advanced* to account for the peculiar paralytic phenomena due to lead poisoning, there is a great variety of opinion. Remak (16) and Vulpian (1) think that a distinct group of cells situated in the chord a little above the cervical swelling is affected by lead, and that through this the paralysis is primarily caused. Friedlaender (7), Leyden, Zunker (8), and others place the primary lesion either in the muscles or in the intramuscular nerve fibers, while some observers, on account of the peculiar galvanic and faradic reaction of the muscles, first described by Eulenberg (18) and Erb (17), place the primary lesion in the nerve itself. This case speaks in favor of the primary lesion taking place in the muscle, but it shows also that the intramuscular nerve fibers and the musculo-spiral nerve undergo degenerative changes, which, if they do not preceed, appear coincidently with those of the muscular structures. The muscle had only parts affected, while the intramuscular nerve fibers were more uniformly and extensively affected, and therefore point to the possibility that the morbid change began in them, and that the myotic changes were merely secondary.

The cases of the authors quoted will be found as references at the end of this paper, as I thought the full history of them would only be interesting to a very few of the members of this Society.

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